Editorial

Environmental Tobacco Smoke: Causative Agent or White Elephant?

THE POSSIBLE EFFECTS of environmental tobacco smoke (ETS) continue to be of concern to researchers, public health administrators, and the general public. In this issue, Roy J. Shepard presents a review of respiratory irritation and ETS. He concludes, primarily on the basis of experimental challenge studies, that ETS causes only small, immediate changes in respiratory function. However, he finds that evidence, derived mainly from epidemiological studies, suggests that these acute changes may progress to chronic reactions and an increased prevalence of chronic respiratory disease. The review is a logical interpretation of the literature, and the conclusions accord with those of previous reviews,[1] the official policy, and public opinion.

Can we see, in spite of this, any clouds on the horizon? When most scientific data are considered, and perhaps particularly data from epidemiological studies, expert opinion may be divided. From a scientific point of view, it is extremely important that such diverse opinions—in this case, on ETS and respiratory disease—continue to be discussed, regardless of whether they support the Environmental Protection Agency, the tobacco industry, or the politician. Only by presenting sound scientific data and continuously scrutinizing its validity can proper public health measures be recommended, evaluated, and revised.

As a toxicologist, I have always been concerned by the rather extensive damage to the lungs attributed in some studies to ETS exposure. In the 1960s, it was demonstrated in animal experiments, by Green et al.[2] and ourselves,[3] that the lung's defense mechanisms are capable of sustaining relatively high doses of inhaled agents—including live bacteria—before damage occurs. Subsequent experiments on tobacco smoke[4] demonstrated that, at low levels of exposure, there may even be a stimulating effect on the immune function. In this perspective, extensive pulmonary effects from the rather low levels of ETS exposure found in some studies can be questioned.

I am also concerned as an epidemiologist. In the literature, a wide range of disparate health consequences, including breast cancer and other diseases unrelated to active smoking, have been associated statistically with ETS exposure.[1] The biological plausibility that ETS causes such a wide variety of effects, and particularly effects not found to be associated with active smoking, must be rather low. Also, for certain diseases, e.g., cardiovascular disease, the increased risk related to ETS is of about the same order as that related to active smoking.

Could it be that we are committing a fundamental error by placing ETS in the category of a causative factor, when in reality we may be studying a confounder? There are two aspects to this issue. First, the general problem of confounding becomes particularly important in studies of weak associations, or low-risk agents such as ETS. This was reviewed extensively by Wynder[5] several years ago. With regard to respiratory infections and ETS exposure, the classic study from Jerusalem[6] demonstrated that social factors were three to four times more important than ETS exposure, although the latter was significantly related to the disease. In all studies on weak associations, the major causative factor must be very precisely described before any certain conclusions concerning less important factors can be drawn—a task that has not been carried out successfully in several of the studies reviewed.

The second aspect relates to new confounders that were unknown at the time of the different studies. There is increasing evidence that dietary habits are related to several kinds of respiratory disease, including lung cancer and chronic bronchitis.[7,8] Evidence also exists that smokers' diets differ from nonsmokers', and there are studies that suggest these differences are present in nonsmoking females, whether married to smokers or nonsmokers.[9] Consumption of fat or smoked foods increases the risk; vegetable and fruit consumption decreases it.

It is too early to conclude how these possible confounders, or any other potential confounders, could influence the conclusions drawn in Shepard's review. It will be interesting, however, to see how well they stand

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up to critical assessment in 5 or 10 y, when results are available from ongoing studies that include dietary habits.

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References

1. The health consequences of involuntary smoking. A report of the Surgeon General. Rockville, Maryland: Department of Health and Human Services. 1986; 1-359.
2. Green GM, Kass EH. Factors influencing the clearance of bacteria by the lung. J Clin Invest 1964; 43:769-76.
3. Rylander R. Pulmonary defense mechanisms to airborne bacteria. Acta Physiol Scand 1968; 306:1-89.
4. Holt P. Immune and inflammatory function in cigarette smokers. Thorax 1987; 42:241-49.
5. Wynder EL, ed. Workshop on guidelines to the epidemiology of weak associations. Prev Med 1987; 16:139-210.
6. Harlap S, Davies AM. Infant admissions to hospital and maternal smoking. Lancet 1974; 1:529-32.
7. Schwartz J, Weiss ST. Dietary factors and their relation to respiratory symptoms. Am J Epidemiol 1990; 132:67-76.
8. Fontham ETH. Protective dietary factors and lung cancer. Int J Epidemiol 1990; 19:32-42.
9. Koo LC, Ho J H-C, Rylander R. Life-history correlation of environmental tobacco smoke. A study on non-smoking husbands. Soc Sci Med 1988; 26:751-60.

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